

LETTERS to the Editor

Vitamin D and Calcium Homeostasis

TO THE EDITOR: We are writing to correct some of the comments made by Dr. H. F. DeLuca in his editorial [Impact of recent findings concerning vitamin D metabolism on clinical medicine. *West J Med* 121:64-65, Jul 1974] concerning our review article "Metabolism and Action of the Hormone Vitamin D—Its Relation to Diseases of Calcium Homeostasis" [*West J Med* 121:22-44, Jul 1974] which also appeared in *THE WESTERN JOURNAL OF MEDICINE*. It is incorrect for DeLuca to state that "vitamin D may be classified as a steroid only because it is derived from a precursor which possesses a steroid nucleus [and that] the resemblance of $1,25-(\text{OH})_2\text{-D}_3$ to a steroid is vague at best." The precursor 7-dehydrocholesterol and both cholecalciferol [vitamin D_3 or technically 9,10-secocholesta-5,7,10(19)-triene-3 β -ol] and $1,25-(\text{OH})_2\text{-D}_3$ [technically 9,10-secocholesta-5,7,10(19)-triene-1 α ,3 β ,25-triol] are all steroids according to the official nomenclature rules of the International Union of Pure and Applied Chemistry (IUPAC).¹ In particular, cholecalciferol and all its known metabolites are "seco" steroids. Seco steroids occur by definition in steroids with the characteristic cyclopentanoperhydrophenanthrene ring structure when "ring fission with addition of a hydrogen atom at each terminal group thus created occurs. These new compounds are indicated by the prefix 'seco,' the original steroid numbering being retained." [See rule 7.4; reference 1.] In fact, the example of a seco steroid given in reference 1 is cholecalciferol.

We certainly stoutly maintain that the mode of action of $1,25-(\text{OH})_2\text{-D}_3$ is analogous to that of many other classical steroids. The reader is directed to the numerous papers published by the laboratories of Lawson and Kodicek, Wasserman, and Norman for a full elaboration of the argument. There is ample documentation of the renal production and distal interaction and localization in the nucleus of the intestinal cells of $1,25-(\text{OH})_2\text{-D}_3$.² This nuclear localization of $1,25-(\text{OH})_2\text{-D}_3$ results in the *de novo* synthesis of a

m-RNA³ which codes for a protein unique to the presence of vitamin D and its metabolite, calcium binding protein.⁴ Certainly these observations qualify $1,25-(\text{OH})_2\text{-D}_3$ as being a steroid hormone of status which is of equal legitimacy to that of estradiol, progesterone, testosterone, etc. However, it is obviously an important problem for all steroids, including $1,25-(\text{OH})_2\text{-D}_3$, to know whether *all* the biological actions occur as a consequence of nuclear interaction. Any reservations DeLuca has in this respect are, of course, equally shared by all workers concerned with the mechanism of action of steroids.

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3. Spencer JD, Emtage DE, Lawson M, et al: The response of the small intestine to vitamin D: Isolation and properties of chick intestinal polyribosomes. *Biochem J* 140:239-247, 1974
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Semi-Retired Physicians

TO THE EDITOR: I have a problem, which I probably share with hundreds of other semi-retired California physicians like myself.

I closed my office three years ago—I am now 75 years of age. My medical work is now mostly done in convalescent hospitals, but not as an employed physician. The number of patients I examine and treat may vary between 30 and 40 a month, which means a medial gross income of just several hundred dollars.

In order to keep up with medical education, especially in Category 1, I am forced to go to the Stanford and San Francisco areas for lectures with